DISSERTATION ON

A CASE-CONTROL STUDY IN CLINICAL SPECTRUM AND RISK FACTORS FOR MYOCARDIAL INFARCTION IN THE YOUNG (≤40 YRS)

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CERTIFICATE

This is to certify that this dissertation titled **"A CASE CONTROL STUDY IN CLINICAL SPECTRUM AND RISK FACTORS FOR MYOCARDIAL INFARCTION IN THE YOUNG (≤ 40 YRS)"** has been prepared by **Dr.S.VENKATESAN** under my supervision in the Department of General Medicine, Chengalpattu Medical College, Chengalpattu during the academic period 2008-2011 and is being submitted to the Tamil Nadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the University regulation for the award of the Degree of M.D. General Medicine and his dissertation is a bonafide work.

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CONTENTS

A CASE – CONTROL STUDY ON CLINICAL SPECTRUM AND RISK FACTORS FOR MYOCARDIAL INFARCTION IN THE YOUNG (≤ 40 YRS)

INTRODUCTION :

Occlusive arterial disease resulting from atherosclerosis is a leading cause of death and disability throughout the world.[1-2]

The global burden of cardiovascular disease carries with it a heavy financial burden[3]

Atherosclerosis represents a chronic inflammatory response to vascular injury caused by a variety of agents that activate or injure endothelium and promote lipoprotein infiltration, retention and modification, combined with inflammatory cell entry.[4-6]

Myocardial Infarction generally occurs with the abrupt decrease in coronary blood flow that follows a thrombotic occlusion of a coronary artery previously narrowed by atherosclerosis. The injury is produced by or facilitated by factors such as cigarette smoking, hypertension, lipid accumulation, Diabetes and a number of other factors.

A quote from Berkeley heart lab – "Just being an Indian descent puts you at a high risk of coronary artery disease". The pattern of coronary artery disease is indeed changing in India. It has been reported to be as follows.

a) Coronary artery disease appears a decade earlier in India than other countries.

b) Males are affected more than females

c) Hypertension and Diabetes account for about 40 percent of all cases.

d) Heavy smoking is an important contributing factor.

The above findings were based on a clinical study done in Chandigarh in persons of age greater than 30[7]

The risk of coronary artery disease in Indians is 3-4 times higher than white Americans, 6 times higher than Chinese and 20 times higher than Japanese.[8]

Indians are prone as a community to coronary artery disease at a much younger age. The disease pattern is severe and diffuse. Premature coronary artery disease is defined as coronary artery disease occurring before the age of 40 years. Coronary artery disease is affecting Indians 5-10 years earlier than other communities. Indians also show higher incidence of hospitalization, morbidity, and mortality than other ethnic groups.[9]

In some studies from India, the percentage of patients below the age of 45 years suffering from AMI is reported as high as 25-40%.[10]

Young patients from other communities do not show extensive disease, whereas in young Indians there is often triple vessel disease with poor prognosis. The post infarction course in also worse in Indians as compared to whites. This is reflected by three time higher rate of reinfarction and two times higher rate of mortaliy.[11]

The prevalence of coronary artery disease according to works done by Destefano F, Merrit RK, Anda RF and Bhatia ML[12] shows that it is two times higher (10%) in urban than in rural India. South Indians have higher prevalence 7% in rural and 14% in urban areas. The vulnerability of urban Indians to

coronary artery disease is possibly related to different nutritional, environmental and life style factors. The BMI in urban Indians as compared to rural Indians is 24 VS 20 in males and 25 VS 20 in females.

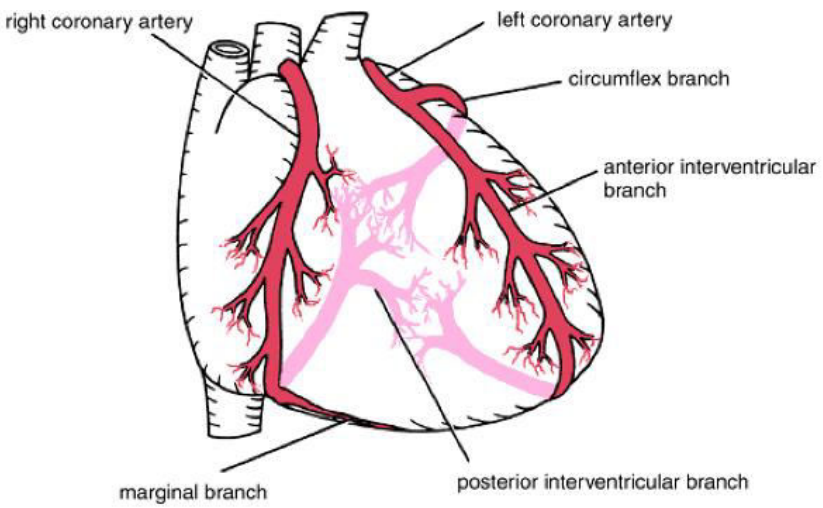
Therefore there has to be a high index of suspicion for coronary artery disease in Indians above the age of thirty. The risk factor evaluation must start earlier. Investigations like tread mill, stress echo, stress thallium and coronary angiography should be more liberally recommended.

The study conducted as a Case-Control retrospective study, aims at evaluating the risk factors involved in development of myocardial infarction and the clinical spectrum in the younger ($\leq$ 40 yrs) age group.

REVIEW OF LITERATURE

The defining characteristic of the coronary circulation is the direct relationship existing between coronary blood flow and myocardial Oxygen consumption. The main parameters dictating cardiac Oxygen consumption are heart rate (chronotopry) contractility (Inotropy), and LV wall stress. Whereas coronary perfusion at rest in human represents about 200ml/min. it can increase up to 1000ml/min on maximal exercise. This difference represents the coronary flow reserve. The mechanism by which the coronary bed adopts blood flow is the cardiac work load represents one component of coronary autoregulation, that is the recruitment of the coronary flow reserve to match coronary flow to energy needs. This is accomplished via 1) Endothelial factors (Nitric Oxide) 2) Metabolic by products (adenosine) and 3) Neural control (both cholinergic and alpha and Beta adrenergic receptors)[13]

ANATOMY OF CORONARY CIRCULATION



The two coronary arteries that supply the myocardium arise from the sinuses behind two of the cusps of the aortic value at the root of the aorta. The

right coronary artery dominance in 70% of individuals and the left has dominance in 10% and shared in 20%[14]

The left anterior descending Coronary Artery	-	Anterior wall of left ventricle, anterior 2/3 of interventricular septum.
Left circumflex coronary Artery	-	Lateral wall of left ventricle
Right Coronary Artery	-	Inferoposterior wall of left ventricle, Post 1/3 of Interventricular septum Right atrium & right ventricle.

PRESSURE GRADIENT AND FLOW IN CORONARY VESSELS

The heart is a muscle that, like skeletal muscle compresses its blood vessels when it contracts. The pressure inside the ventricle is slightly higher than in the aorta in systole, hence flow occurs in the arteries supplying the subendocardial portion of the left ventricle only during diastole. Because there is no blood supply during systole in the subendocardial portion of the left ventricle, this region is prone to ischemic damage and is the most common site of Myocardial Infarction. On the other hand as the right ventricle pressure difference with aorta is greater, the flow in right coronary artery is not reduced in systole.[13]

CORONARY ANASTOMOSIS?

Anastomosis between branches of coronary arteries, subepicardial or myocardial and between arteries and extra cardiac vessels are of prime importance. Clinical experience suggests that anastomosis cannot rapidly provide collateral routes sufficient to circumvent sudden coronary obstruction. It is hence traditional to regard coronary arteries as end arterial.

MIOCARDIAL INFARCTION DUE TO CORONARY ATHEROSCLEROSIS

Myocardial infarction generally occurs with the abrupt decrease in Coronary blood flow that follows a thrombotic occlusion of a coronary artery previously narrowed by atherosclerosis.

CORONARY ATHEROSCLEROSIS

The earliest lesions of atherosclerosis can be found in young children and infants in the form of fatty streak whereas the advanced lesion, the fibrous plaques, generally appears during early adulthood and progress with age.

The abdominal aorta is involved earliest. The aorta is usually most heavily involved at or near the orifice of its branches (particularly at the level of coronary arteries)

In coronary arteries, raised lesions are most prominent in the main stems, the highest incidence being a short distance beyond the ostia. Atherosclerosis is usually always found in the epicardial (extramural) portions of the vessels, while the intramural coronary arteries are spared coronary atherosclerosis is often diffuse. The degree to which the lumen is narrowed varies, but once the process has commenced, all the intima of the extramural portions of the vessels is usually involved. Typical atheromatous fibrous plaques also develop in saphenous vein, which is used for aortocoronary by pass graft.

HYPOTHESES OF ATHEROSCLEROSIS

- **The response to Injury Hypothesis**

The endothelial lining cells are exposed to repeated or continous insults. Dysfunctional endothelial cells at the susceptible sites in the arterial tree would lead to exposure of the subendothelial tissue to increased concentrations of plasma constituents. This triggers a sequence of events including monocytes and platelet adherence, migration, platelet aggregation and formation of microthrombi and release of secretory products. This causes proliferation of smooth muscle cells at these sites of injury. Monocytes become transformed to foam cells. Thus a well developed plaque is formed.

- **Monoclonal hypothesis**

This states that the intimal proliferative lesion results from the multiplication of single, individual smooth muscle cells, as do benign tumors. According to this hypothesis, the intimal smooth muscle cell that proliferate to form an atheroma are normally under feedback control by mitosis inhibitors formed by the smooth muscle cells in the contiguous media, and this feedback control system tends to fail with age as these controlling cells die and are not adequately replaced.

RISK FACTORS

A number of conditions and habits are present more frequently in individuals who develop atherosclerosis than in general population ; these factors have been termed risk factors.

The risk factor concept implies that a person with at least one risk factor is more likely to develop a clinical atherosclerotic event and is likely to do so earlier than a person with no risk factors. The presence of multiple risk factors further accelerates atherosclerosis.

Hypercholesterolemia, hypertension and cigarette smoking may be the most potent factors for atherosclerosis. Risk factors also vary in terms of their potential reversibility with current techniques of preventive management.

The American College of Cardiology's Bethesda Conferences placed risk factors into four categories according to the likelihood that modification of the factor will result in lower risk.

These categories adopted from the 27th Bethesda Conference are as follows:

CATEGORY I

- Basic research and human observational study indicate a clear causal relationship.
- Intervention data demonstrates the magnitude of the benefit and risk.
- Interventions are cost-effective and have been proved to lower coronary artery disease risk.

1. Cigarette smoking
2. LDL Cholesterol
3. High fat/high cholesterol diet
4. Hypertension
5. Left Ventricular Hypertrophy

CATEGORY II

- Studies indicate a casual relationship.
- Intervention data for large scale trials are limited.
- Cost effectiveness not ascertained but likely to lower coronary artery disease.

1. Diabetes Mellitus
2. Physical inactivity
3. HDL Cholesterol
4. Triglycerides, ; small, dense LDL
5. Obesity
6. Post menopausal status of women

CATEGORY III

- Studies indicate association.
- Interventions have not been tested adequately.
- Might lower risk.

1. Psychosocial factors
2. Lipoprotein (a)
3. Homocysteine
4. Oxidative stress
5. No alcohol consumption.

CATEGORY IV

- Factors associated with coronary artery disease Risk, but cannot be modified

    1. Age
    2. Male gender
    3. Low socio economic status
    4. Family history of early onset of coronary artery disease.

SMOKING

A strong dose response relationship between cigarette smoking and Coronary Heart Disease has been observed in the young, in the elderly and in all recial groups.[15]

Cigarette smoking increases risk 2 to 3 fold and interacts other risk factors to multiply risk. Fitters and modification won't reduce risk. Pipe smoking and cigar smoking increase the risk of Coronary Heart Disease

More than 1 in every 10 Cardiovascular death in the world in the year 2000 were attributable to smoking.[16]

Exposure to tobacco smoke by non smokers was consistently associated with a 20 to 30% increase in risk.[17]

Oxidative stress plays a central role in smoking mediated dysfunction of nitric oxide biosynthesis in endothelial cells. Smoking also lower HDL – C. These effects along with direct effects of Carbonmonoxide, nicotine, produce endothelial damage. Smokers have increased vascular reactivity, reduced Oxygen Carrying capacity, lower threshold for myocardial infarction and

increased risk of coronary spasm. Also associated with increased levels of fibrinogen and increased platelet aggregability.

In a previous smoker the relative risk decline nearly to that of nonsmoker in a year or less.[18]

Cessation of smoking was found to reduce Coronary Heart Disease mortality by 36%. This reduction does not change with age, gender, country.[19]

HYPERTENSION

Both systolic and diastolic Hypertension have strong, positive, continuous, and graded relationship to Coronary Heart Disease with out evidence of threshold risk level of Blood Pressure.[20]

A widened pulse pressure an indicator of arterial stiffness is another Blood Pressure parameter that predicts Coronary Heart Disease.[21]

Mechanism by which Hypertension may cause coronary events include impaired endothelial function, increased endothelial permeability to lipoproteins, increased adherence of leukocytes, increased oxidative stress, Hemodynamic stress triggering acute plaque rupture, and increased myocardial wall stress and oxygen demand.[22]

In an analysis of 354 randomized trials regimens of multiple drugs given at lower dose were estimated capable of reducing systolic BP by 20mm Hg and Diastolic BP by 11 mm Hg effects that could result in stroke reduction of 63% and Coronary Heart Disease of 46%.[23]

In 2000 the estimated number of adults with Hypertension was 972 million, 333 million from developed countries, 639 million from developing

countries by 2025 the total number of adults with Hypertension is anticipated to exceed 1.5 billion.[24]

HYPERLIPIDIMIA

Both hypercholesterolemia and hypertriglyceridemia are important factors for coronary artery disease. A 10 percent increases in serum cholesterol is associated with a 20 to 30 percent increase in risk of coronary artery disease and elevations earlier in life may be associated with higher increases in risk.

The increases in cholesterol are associated mainly with a raise in LDL concentrations; the increases in triglycerides are associated with a raise in VLDL and remnants of their catabolism (mainly IDL). Metabolic mechanisms have been postulated whereby abdominal obesity, which is associated with insulin resistance of peripheral tissues and compensatory hyperinsulinemia, promotes enhanced production of triglyceride and cholesterol rich lipoproteins by the liver and consequent hyperlipidemia, hypertension and hyperglycemia (the insulin resistance syndrome)

This is associated unequivocally with increased incidence of premature ischemic heart disease. In Framingham study cholesterol levels in men below age 40 were closely related to the future development of ischemic heart disease. This relation was less pronounced in older age group. In multiple Risk Factor Intervention Trial men with cholesterol above 240 mg/dl had more than a threefold increase in risk of ischemic heart disease death compared with men with cholesterol below 200 mg/dl.

This may be associated with premature atherosclerosis in some specific disorders; this association increases the risk of diabetics, hypertensive and smokers.

According to studies by Moorjani S et al, in Indian patients with coronary artery disease high triglyceride levels are found more often than high cholesterol levels. Triglycerides bring change in LDL particle size, density, distribution and composition producing smaller, denser, and more atherogenic particles.

Studies by Jeppesen J, Hein H, suadiciani P, state that an increase of triglycerides by 90mg/dl has the same effect on coronary atherosclerosis, as increase in age by 10 years.

Earlier there has been an under emphasis on the significance of triglycerides as risk factor for coronary artery disease. Indians worldwide have demonstrated a traid of high triglycerides with high LDL-C levels and low HDL levels.

Studies by Kishore S et al[25] state that higher level of apolipoprotein B is reported in 1/3 of Indian males. This factor in combination with low levels of HDL and Hypertriglyceridaemia result in formation of small dense LDL. This increases the risk of coronary artery disease more than three times.

The LDL – cholesterol types are described as phenotypes A, B or C, which are genetically determined. Patients with LDL – phenotype B have predominantly small and dense LDL particle which constitute an important risk factor for coronary artery disease. A 75% prevalence of phenotype B is seen in

Asian Indians in contrast to 25% in white population[26] (Enas EA, Salim Yusuf).

Clear benefits have been demonstrated for dietary and pharmacological treatments that lower serum cholesterol. Treatment aimed at lowering serum cholesterol by 10 percent reduces the risk of coronary artery disease by 15 percent.

DIABETES AND INSULIN RESISTANCE

Insulin resistance and Diabetes mellitus reach among the major Cardiovascular risk factors.

Patient with Diabetes have 2 – 8 fold of high risk of developing Coronary Heart Disease as compared with Age and ethinically matched non Diabetic individuals and 75% of all death in Diabetic patients is associated with Coronary Heart Disease.

Insulin resistance alone confers an elevated risk of Congestive Heart Failure and probably explain the association of Obesity it is common vascular event.[27]

Diabetes abolish the usual protection from Coronary Heart Disease afforded a premenopausal women. Diabetic women have twice the risk of recurrent myocardial infarction compared with Diabetic men.[28]

Mechanism by which Diabetic cause atherosclerosis included low HDL-C, high Triglycerides, increased lipoprotein remnants, increased small density LDL – C, elevated lipoprotein A, increased platelet aggregability, impaired endothelial function.

As recently demonstrated in the DM control and complication trial, intensive treatment of Diabetes over 6 year period reduced the risk of CHD by 42%.[29]

OBESITY

Obesity is defined by the AHA as a major risk factor for Coronary Heart Disease. Obesity is associated with insulin resistance, hyperinsulinemia, Type 2 DM, Hypertension, Low HDL – C, small dense LDL, inflammation and elevated CRP, LVH.[30] In women, obesity contributes independently from physical activity to the development of Coronary Heart Disease.[31]

Obesity accelerates the progression of coronary atherosclerosis in adolescent and young adult men.[32]

Body mass index (BMI) has been adopted widely as a measure of adiposity. BMI is calculated as weight (kg) / Height (m^2)

Normal Weight	-	BMI of 18.5 to 24.9
Over Weight	-	BMI of 25 to 29.9
Obesity	-	BMI $\geq$ 30

BMI correlates with total body fat content. In multivariate analysis – obesity is not usually found to be an independent risk factor. Nevertheless some large prospective observational studies indicate that obesity is an independent risk factor in men and women.[33]

Weight loss improves insulin sensitivity and glucose disposal, reduces HBA1C in patients with Type 2 DM, reduces BP and Triglycerides, produces a modest reduction in LDL-C, and increases HDL-C.

PHYSICAL INACTIVITY

Physical inactivity is an independent risk factor for Coronary Heart Disease and roughly doubles the risk. Physical inactivity slows the progression of angiographycally defined coronary atheroscleorsis in human.[34]

Morris and colleagues first reported in the mid-1950 that coronary artery disease rates were lower in people who were involved in various physical activities than those with sedentary life style.

Moderate intensity physical activity reduce the coronary heart disease risk by decreasing myocardial oxygen demand, increased myocardial efficiency and electrical stability, increased HDL – C, decreasing BP, reducing Obesity, improving insulin secretion.

The risk of coronary artery disease in sedentary individuals was almost twice that of active individuals after controlling for other coronary risk factors. Long-term prospective studies of men and women consistently demonstrate that regular physical activity protects against death from coronary artery disease. These benefits apply to activities as simple as brisk walking which has been shown to reduce the risk of coronary artery disease in women and men as well as the risk of type II diabetes.[35]

WAIST – HIP RATIO

Waist circumference is the minimum circumference measured between costal margin and iliac crest and hip circumference is measured over the buttocks waist hip circumference ratio more than 0.83 in females and 0.93 in males were taken as abnormal.

WHO has adopted BMI $\geq$ 25 as over weight. Studies by Diwivedi shows that 80.35% males and 91.66% females were centrally obese, 27.5% females who had BMI less than 25 manifested upper segment obesity. Thus waist hip ratio is of great significance.

PSYCHO SOCIAL FACTORS

Psychosocial factors such as depression, absence of social support and anger appear to contribute to an elevated risk of coronary artery disease although further data are needed to confirm these relationships and establish the efficacy of interventional strategies.

When the personality characteristics of coronary artery disease patients are examined, it is found that there is preponderance of certain type of personality traits, known collectively as coronary prone type A behaviour (Friedman & Roseman).

The type A behaviour is characterized by

1. Time Urgency.
2. Excessive Competitiveness and hostility

Overall, there is a chronic struggle to achieve or complete a large number of tasks, working against the limits of time available.

In contrast type B personality is just the opposite, characterized by a relaxed unhurried attitude and less vigorous attempts to achieve a goal.

Studies of therapeutic interventions suggest a role for improving psychosocial factors as part of preventive programs, particularly in secondary

prevention. The strongest evidence comes from post myocardial infarction patients.

A recent Meta analysis of 37 small studies of health education and stress management programs for coronary artery disease patients suggested that such efforts might reduce cardiac mortality by 35% and recurrent myocardial infarction by 29%. This is due to favourable effects on blood pressure, cholesterol, body weight, smoking behaviour, physical activity and dietary habits.

LIPOPROTEIN A

The normal function of Lp(a) (Lipoprotein a) is unknown, However Lp (a) may inhibit endogenous fibrinolysis by competing with plasminogen for binding on endothelial surface and increase the release of PAI.

Lipoprotein-a is now recognized as an independent risk factor for coronary artery disease. It is a genetic risk factor. It is not affected by life style modifications like changes in diet and exercise. Lp-a is ten times more atherogenic than LDL-C.

Lp-a values are more or les constant in a single individual since birth and hence estimation of Lp(a) once in a life time irrespective of age is enough for cardio vascular risk assessment.

According to studies by Dilip Kr. Mukherjee et al Lp(a) values were higher amongst both Indian boys and girls and more so amongst girls compared to boys.

Lipoprotein – Level in Boys	19.38 ± 5.08 mg %
Girls	27.26 ± 4.02 mg %

Lp(a) promotes early atherosclerosis and thrombosis. Lp(a) is a stronger risk factor than DM for coronary artery disease in younger women. In Indians, both in India and abroad, the levels of Lp(a) are higher as compared to the whites, suggesting a genetic propensity (Dwington PN et al).

Lp-a levels according to Sahan et al of cord blood are higher among Indian newborns than Chinese newborns and this difference is also associated with a fourfold higher coronary artery disease – related mortality in Indians than Chinese in Singapore.

Hunt S et al in his studies state that Lp-a levels over 40 mg/dl increases the risk associated with cigarette smoking by 19 times, with DM by 3.4 times, with high total cholesterol by 4.2 times, with hypertension by 4.6 times, with high TC/HDL ratio by 6.9 times and with high homocysteinemia by 9.3 times.

Young individuals who have suffered infarction yet who appear to lack any traditional risk factors are more suitable for assessment of Lipoprotein a as this would be the contributing factor.

HOMOCYSTEINE

Homocysteine is a highly reactive sulfur containing amino acid that is an intermediate product of methionine metabolism Patients with rare inherited defects of methionine metabolism can develop severe hyperhomocystinemia (Plasma levels > 100 micromol/lt) and can have premature atherothrombosis.[36]

The mechanisms that account for these effects remain uncertain but may include endothelial toxicity accelerated oxidation of LDL-C, impairment of Endothelial derived relaxing factor and reduced flow mediated arterial vasodilatation.

Mild to moderate levels of homocysteine (Plasma level>15 micromol/l) are common in general populations, primarily due to insufficient dietary intake of folic acid, vitamin B_{12} other patient groups that tend to have elevated homocysteine levels include smoking, alcohol, those with increased Age, male sex, menopause common polymorphisms in the methyltetra hydrofolate reductase gene, those receiving folate antagonists such as methotextrate, and carbamazepine and those with impaired homocysteine metabolism due to hypothyroidism or to renal insufficiency.

A large series of cross sectional and retrospective studies and a meta analysis of 27 observational studies indicate a positive relationship between mild to moderate hyperhomocysteinemia and atherosclerosis.[37]

A large cohart study from Norway reported a relative risk of CHD of 1.4 for every 4 micromole / L. increase in plasma homocysteine.[38]

However because homocysteine levels increase after myocardial infarction and stroke such data cannot be used to establish a cause and effect relationship.

Two large clinical randomized placebo controlled Trials in high risk subject found no reduction Cardiovascular events from supplementation with folate and Vit B_6, B_{12} , but they shown reduction in Homocysteine level.

OXIDATIVE STRESS :

Oxidative modification of LDL – has been hypothesized to play a major role in the initiation of progression of Atherosclerosis. Naturally occurring anti oxidents are Vit E, C & betacarotene.

Basic research suggests that Vit E may delay or prevent various steps in atherosclerosis. However randomized trial data are not yet sufficient to fully assess the role of vitamin E and Vitamin C or other antioxidants in the primary or secondary prevention of atherosclerotic disease.

The evidence available does not establish that vitamin E and reduces the risk of coronary artery disease.[39] Longer follow up of the completed trials, as well as ongoing trials, will provide valuable information upon which rational clinical decision for individuals and policy for health of the general public can be reliably based. Although it remains unclear whether antioxidant supplementation will reduce the risk of chronic disease, consumption of fruits and vegetables high in these micronutrients is an important part of healthy diet.

ALCOHOL INTAKE :

Heavy Alcohol intake is associated with increased the risk of death from several cause and is a major public health concern. However cross sectional case control and cohart studies indicate that mild to moderate alcohol intake is associated with reduced rates of CHD compared with no alcohol intake. These studies suggest a "J" shaped relationship between the level of alcohol intake and total mortality.[40]

High alcohol intake is associated with an increased risk of high blood pressure. It appears that alcohol consumption raises systolic pressure more

than diastolic. But the finding that blood pressure returns to normal with abstinence suggests that alcohol induced elevations may not be fixed, and do not necessarily lead to sustained blood pressure elevation.

A large Danish population based cohart, women who drug atleast once a week had lower risk of CHD than women who drawn less frequently although more frequent alcohol consumption did not result in additional risk reduction.[41]

Alcohol intake increase total HDL level as well as HDL_2, HDL_3 B also has potential beneficial effects on fibrinolytic function, platelet aggregation, inflammation, endothelial function.

AGE

Myocardial Infarction risk increases with age. In the age group less than 40 years highest incidence is in the age of 35 – 40 years. Ischemic heart disease is the major cause of death in males > 35 and all patients > 45.

This can be attributed to the fact that as age increases in both sexes and the blood pressure also increases this may also be due to accumulation of environmental toxins.

SEX

Atherosclerotic involvement of the coronary arteries is well established in man by young adulthood.[42]

Females are generally more protected from myocardial infarction than men. Women seen with a first myocardial infarction tend to be older than men with first myocardial infarction. The gap narrows substantially after menopause.

In most studies of young myocardial infarction < 40 years 90% of the cases were male. In females coronary artery disease started 10 years later than men and the incidence raised more towards the menopausal age group.

POSTMENOPAUSAL STATUS

Coronary heart disease is relatively uncommon in premenopausal women but men exhibit a higher incidence of coronary artery disease at early age, as well as higher mortality rates from it, the gap narrows substantially after both natural menopause and bilateral oophorectomy. Women seen with a first myocardial infarction tend to be older and have higher mortality than men with first myocardial infarction.

A wide range of factors may explain the increased risk of coronary artery disease after menopause, including adverse changes in lipid and glucose metabolism that result in an increase in LDL cholesterol and a decrease in HDL cholesterol, an increase in glucose intolerance and changes in haemostatic factors and vascular function. Endogenous estrogens appear to play a major role in reducing the risk of coronary artery disease in women. There is also a protective effect of vascular function as well as an apparent estrogen related protection of LDL from oxidation. Estrogen also plays one or more roles in maintaining normal haemostasis and improving glucose tolerance but recent studies showing Hormone replacement Therapy fails to demonstrate the benefit indeed showing increased incidence of Cardiovascular events, ca brest, Thromboembolism.[43]

FAMILY HISTORY & GENETIC FACTOR

More than 35 case control and prospective studies have consistent identified an association between CHD and a History of 1st degree relatives with early onset CHD.

Ischemic heart disease is well recognized as clustering in families. At present 40% of risk of developing ischemic heart disease is contributed by genetic factors and 60% by environmental factors.

Recent studies have identified a common deletion polymorphism of ACE gene which is associated with increased level of ACE and risk of coronary artery disease. Genetic markers such as LDL receptor, factor V leiden are also other genetic markers. Genetic factors may play a role by ascertaining direct effects on the arterial cell structure and metabolism as they may act indirectly via such factors as hypertension, increase LDL, diabetes mellitus, obesity, Lipoprotein-a, etc.

Coronary artery disease affects Indians 5-10 years earlier than other communities. There is a parallel corollary between coronary artery disease in Indians and the malignant course of rheumatic fever, rheumatic heart disease with associated severe pulmonary hypertension observed by Indian cardiologists. Siblings and children of early CHD patients should be screened for CHD risk factors.

DIET

Diet is an important component of any prevention program in as much as weight reduction can improve dyslipidemia, hypertension and diabetes. One of the most consistent finding in dietary research is that individuals who

consume higher amounts of fresh fruits and vegetables have lower rates of heart disease and stroke. Two randomized clinical trials of dietary inventions showed that the risk of cardiac death or acute myocardial infarction was 65% lower. Low fat diet has been shown to reduce the risk of myocardial infarction in healthy individuals and may even cause regression of coronary artery disease. In the opposite direction, saturated and transfatty acids appear to increase the coronary artery disease. Antioxidant, vitamins, folate supplement, whole grains, fiber, fish and fish oil seems to have a beneficial effect.

The WHO expert committee has given the following dietary changes to be appropriate for high incidence populations.

1. Reduction of fat intake to 20-30 percent of total energy intake.
2. Consumption of saturated fats must be limited to less than 10% of total energy intake: some of the reduction in saturated fat may be made up of mono and poly unsaturated fats.
3. A reduction of dietary cholesterol to below 100 mg per 1000 k cal/day
4. An increase in complex carbohydrate consumption (vegetables, fruits, whole grains and legumes).
5. Reduction of salt intake to 5g daily or less.

Thus a healthy diet can considerably lower the risk of myocardial infarction.

FIBRINOGEN

Several studies have shown that plasma fibrinogen level predicts the future risk of MI and stroke. This indicate that individual with fibrinogen concentration of in the upper third of the control distribution have a relative risk of future Cardio Vascular Disease 2.0 to 2.5 times of that individuals with lower level.[44]

Plasma fibrinogen critically influences platelet aggregation and blood viscosity, interacts with plasminogen binding and in combination with thrombin mediates the final step in clot formation. In addition, fibrinogen associates positively with age, obesity, smoking, diabetes, and LDL-C and inversely with HDL-C, alcohol use physical activity and exercise level.[45]

Despite major genetic determinants of fibrinogen concentration, substantial, variation in plasma levels result from environmental factors. Smoking cessation increased exercise and weight loss can reduce fibrinogen concentration. Fibric acid derivatives also reduce fibrinogen, apparently through PPAR-a mechanism.

MARKERS OF INFLAMMATION PREDICT FUTURE RISK

These markers include nonspecific acute phase remnants such as hs-CRP, adhesion molecules such as ICAM-1 that are involved in mononuclear cell attachment to the vascular endothelium and cytokines such as IL-6 and Tumor necrosis factor. A large body of consistent evidence validates use of acute phase reactants such as CRP and serum amyloid A as markers of risk. Serum amyloid A can bind to HDL particles perhaps rendering them less protective against vascular inflammation.

Single, non fasting measure of CRP a potent predictor of first cardiovascular event among man, women, elderly. Those with metabolic syndrome, Dm, and smokers.[46]

CRP increased in all the CHD risk factors, and decreased with moderate alcohol intake, physical activity, wt loss, and drugs like statins, fibrats, niacin ARB.[47]

hs – CRP will likely prove the most clinically useful because it is easy and inexpensive to measure with commercial assay. hs-CRP increases the relative risks of future event of coronary artery disease 3 to 4 times higher than those with lower hs CRP levels.

INJECTION

Chronic infection with agents such as chlamydia pneumonia, helicobacter pylori, herpes simplex virus or cytomegalovirus virus can lead to systemic inflammation. Such observations have heightened interest in the hypothesis that infection may contribute to coronary risk. Several cross sectional and retrospective studies have identified chlamydia species as well as viral particles in atheromatous lesions. It is important to recognize several mechanisms by which infection might contribute to plague instability. Eg. Chlamydia species have been reported to induce macrophage foam cell formation and increase procoagulant activity human atneromas often contain chlamydia heat shock protein 60, an effector of activation of macrophages, endothelium and matrix metalloproteinase expression.

MINOR RISK FACTORS

Copper deficiency and zinc excess – This leads to secondary hypercholesteremia leading to increased risk of myocardial infarction.

Water hardness – Water hardness is inversely related to ischemic heart disease. Soft water is generally associated with greater risk of heart disease. Metals like magnesium chromium selenium and zinc have protective effect against myocardial infarction. Cadmium, Manganese and Lead have harmful effects and increase risk for myocardial infarction.

Heavy coffee drinking – Produce tachycardia, arrhythmia and extra systole and thus predispose individuals to increased risk of myocardial infarction. Lipid rich fractions from boiled coffee increases serum cholesterol level thus increasing the risk for myocardial infarction.

Blood Group – Persons with type O blood group appear to be at a lesser risk for myocardial infarction. Research is still on going to find out the correlation of such an association.

CLINICAL SPECTRUM OF MYOCARDIAL INFARCTION

SYMPTOMS

In up to one half of cases, a precipitating factors appears to be present before AMI, such as vigorous physical exercise, emotional stress, or medical or surgical emergency illness. Although MI can present at any time of the day, circadian variation have been reported such as clusters are seen in the morning with in few hours of awakening.

- Pain in most common presentation.
- Pain is deep and visceral: adjuncts commonly used are heavy, squeezing, crushing occasionally stabbing or burning.
- Accompanying symptoms are weakness, sweating, nausea, vomiting, anxiety, sense of in pending doom.

Atypical presentation

- Acute confusion (elderly)
- Profound weakness

EXAMINATION

Signs of sympathetic activation – Pallor, sweating, tachycardia

Signs of Vegal activation – Vomiting bradycardia

Signs of Impaired Myocardial function– hypotension, oliguria, cold peripheries, narrow pulse pressure, raised JVP, third heart sound, quiet first heart sould, diffuse apical impulse and lung crepitations.

Signs of tissue damage – fever,

Signs of complications – eg. Mitral regurgitation, pericarditis

Laboratory Findings

The laboratory tests of value in confirming the diagnosis of myocardial infarction are divided into four groups.

1. The electrocardiogram.
2. serum enzyme changes
3. Cardiac imaging.
4. non specific indexes of tissue necrosis and inflamation

Non Specific Reactions

Polymorphonuclear Leukocytosis appears within hours, lasts for 3 to 7 days;

ESR increases – Peaks at first week and remain elevated for 1 to 2 weeks.

ELECTRO CARDIOGRAPHY

Earliest ECG changes are usually ST elevation. Later on there is diminution in the size of R wave and in Transmural (full thickness) infarction – Q wave develops. Subsequently T wave becomes inverted because of change in ventricular repolarisation.

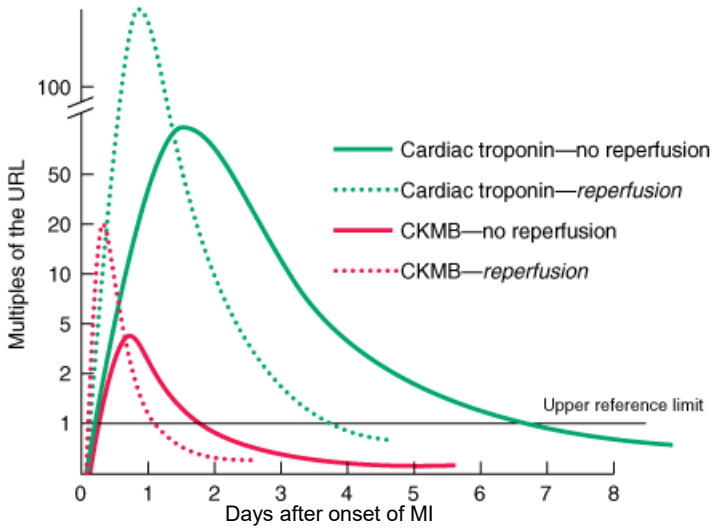
In subendocardial infarction there is ST/T wave changes without Q waves or prominent ST elevation often accompanied by some loss of R waves in the leads facing the infarct.

Anteroseptal infarction	$V_1 – V_4$
Anterolateral infarction	Lead I, aVL, $V_4 – V_5$
Extensive anterior wall infarction	Lead I, aVL, $V_1 – V_6$
Inferior infarction	II, III and aVF
Posterior wall infarction	Reciprocal changes of ST depression and tall R wave in leads $V_1 – V_2$
Right Ventricular Infarction	V_4R – ST Segment Elevations.

Bio Markers

Certain proteins, called serum cardiac biomarkers, are related from necrotic heart muscle after STEMI. The rate of liberation of specific protein

differs depending on their intracellular location, molecular wt, local blood and lymphatic flow.



Tpoponin 1 and II

More specific, are released in 4 to 6 hrs peak at 1 – 2 days remain elevated upto 7 days, So not useful in reinfant.

CPK MB :

Reduced with in 4 to 8 hrs return to Normal in 48 – 72 hrs so useful in reinfarction, drawback in non-specific.

AST starts to rise about 12 hours after infarction and reaches a peak on first or second day, returning to normal within 3 to 4 days.

LDH starts to rise after 12 hours reaches a peak after 2 or 3 days and may remain elevated for a week more.

CHEST X-RAY

This demonstrates signs to left ventricular failure, cardiomegaly and coexisting cardiac or pericardial diseases.

ECHO

Early deduction of wall motion abnormalities can aid in management decision and estimation of the Left Ventricular function is useful prognostically, useful to detect in complications such as cardiac rupture, Ventricular septal defect, pericardial effusion.

RADIONEUCLOTIDE SCANNING – OTHER IMAGING MODALITIES

Isotope used are 201_{Ti} or $99m_{Tc}$ – sestambi. In the presence of fixed coronary stenosis there is an inability to increase myocardial perfusion in the territory supplied by the stenotic artery creating a flow difference and inhomogeneous distribution of isotope. Extremly sensitive in distinguishing acute infarct from chronic scar.

CARDIC CATHETERIZATION AND ANGIOGRAPHY

These remain the only technique that can define coronary anatomy with sufficient precision to support decisions regarding coronary surgery or catheter based interventions in patients with coronary artery disease.

MANAGEMENT

Early management

- Bed rest
- High flow oxygen if $Spo_2 < 90\%$
- Oral Aspirin, Clopidogrel, Nitroglycerine, atorvastatin
- IV access
- IV analgesic with opiates
- IV antiemetic

- Thrombolysis with IV streptokinase
- IV β adrenoreceptor antagonist
- ACE Inhibitors
- Continuous cardiac monitoring and ECG
- Detect and treat complications early

Coronary thrombolysis helps restore coronary patency, preserves left ventricular function and improves survival. Successful thrombolysis leads to reperfusion with relief of pain, resolution of acute ECG changes and sometimes transient arrhythmias. The sooner the patient is treated the better the results.

Clinical trials have shown that appropriate use of these drugs can reduce the hospital mortality of myocardial infarction by between 25% and 50% short term mortality was reduced from 13% to 8%

Streptokinase 1.5 Million units in 100 ml saline IV over 1 hour is used. It is antigenic and occasionally causes serious allergic manifestation. The drug may cause hypotension and is antigenic. Alteplase is not antigenic and seldom causes hypotension.

ABSOLUTE CONTRAINDICATIONS FOR THROMBOLYSIS

- Uncontrolled hypertension (>180/110)
- Active internal bleeding
- Previous arachnoid or intracerebral hemorrhage
- Ischemic stroke in past 1 ‘yr’ (s)
- Aortic dissection.

RELATIVE CONTRAINDICATIONS FOR THROMBOLYSIS

- Anticoagulant with INR ≥ 2
- Recent surgery (with in 1 month)
- Known bleeding diathesis
- Active peptic ulcer disease
- Pregnancy
- Hemorrhagic diabetic retinopathy
- 5 days to 2 years of recent streptokinase

ANGIOPLASTY

Immediate or primary angioplasty of the infarct – related coronary artery is relatively safe and effective alternative to thrombolytic therapy

ANTICOAGULANTS

Unfractioned heparin bolous of 60U/kg followed by initial infusion of 12U/kg/ hr in addition to reinfarction after successful thrombolysis, reduce the risk of thromboembolic complications. Clinical trials have shown that this form of therapy produces a small reduction in short term mortality but also increases the risk of cerebral hemorrhage and of other bleeding complications.

βBLOCKERS, NITRATES AND OTHER AGENTS

Acute β adrenoreceptor antagonist use with IV metaprolol relieves pain, reduces arrthymias and improves short term mortality in patients who present within 12 hours of onset of symptoms, but should be avoided if there is heart failure, heart block or severe bradycardia.

Sublingual glyceryl trinitrate is a valuable first aid measure in threatened infarction and IV nitrates are useful for left ventricular failure and the relief of recurrent or persistent ischemic pain.

AGE INHIBITORS

ACE Inhibitors should be prescribed in all hoemodynamically stable patients it reduces the ventricular remodeling with subsequent reduction in the risk of left heart failure. It should be prescribed within 24 hours.

COMPLICATIONS OF MYOCARDIAL INFARCTION

- Ventricular Dysfunction
- Cardiogenic Shock
- Right Ventricular Myocardial Infarction
- Mechanical Causes of Heart Failure

  Free wall rupture

  VSD

  MR
- Arrythmias

  1. Ventricular premature beat
  2. Ventricular tachycardial/fibrillation
  3. Accelerated Idio ventricular rhythm
  4. Supraventricullar arrythmias
  5. Sinus bradycardia
  6. Atrioventricular conduction disturbances
  7. Asystole

- Other complications

    1. Recurrent chest discomfort
    2. Pericarditis, pericardial effusion
    3. Thromboembolism
    4. Left Vetricular anerurysym
    5. Dressler's Syndrome

POST INFARCTION RISK STRATIFICATION AND MANAGEMENT

In stable patients sub maximal exercise stress testing should be carried out prior to hospital discharge to detect residual ischemia and ventricular ectopy, and to provide the patient with a guideline for exercise in early recovery period. Alternatively, or in addition a maximal exercise stress test may be carried out 4 to 6 weeks following infarction. Evaluation of left ventricular function at rest and during exercise is usually warranted as well recognition of a depressed left ventricular ejection fraction by ECHO or radioneucleotide ventriculography helps the physician with the selection of pharmacologic measures to improve long term outcome. Patients with angina induced at relatively low work loads, those who have a large reversible defect on perfusion imaging or a depressed ejection fraction and demonstrate ischemia, and those in whom exercise provokes symptomatic ventricular arrhythmias should be considered at high risk for recurrent myocardial infarction or death from arrythmia and cardiac catherisation with coronary angiography or invasive eletrophysiologic evaluation.

A variety of secondary preventive measures are at least partially responsible for improved long term mortality and morbidity following

myocardial infarction. The benfits and use of beta blockers, antiplatelet agents and anticoagulants and ACE inhibitors are discussed above. Finally risk factors for atherosclerosis should be discussed with patient. In particular efforts should be made to ensure the cessation of smoking and the control of hypertension and hyperlipidemia. Additionally regular physical exercise and reduction of emotional stress should be encouraged.

RISK FACTOR ANALYSIS

Studies by Kaul et al.[48] showed data about the risk factors for MI. Smoking is the most common risk factor.

Smoking	-	76.2%
Hyperlypidemia	-	36%
Hypertension	-	32%
Family history	-	28%
Diabetis Mellitus	-	5%
Multiple risk factor	-	56%

Fourmers propsective study[49] on young MI showed smoking (78%) the most common risk factor.

Study conducted in Singapore[50] by PA Tambyah et al. showed smoking (84%) the most common risk factor.

Ht	19%
FH	20%
Smoking	84%
DM	16%
Hyper cholesterolemia	56%

Dolder M. et al.[51] done a study in 9 countries showed smoking (80%) the most common risk factor.

Studies from South African[52] journal showed smoking (79%) and hypercholesterolemia (52%) the common risk factor.

Nitter hauge et al B53 did a study on 66 patients about risk factors the studies revealed, that smoking was present in 86%

Smoking	86%
Hypercholesterolemia	35%
Systolic hypertension	24%
Diabetes Mellitus	Nil

PK Biswas A. Dasbiswas et al (1994) B54 study on Risk factor and angiographic profile of coronary artery disease in young showed that smokers and hyperlipidemics were the most affected.

PK BISWAS A. DASBISWAS STUDY

Smokers > 10 Cigarettes/day	56.4%
Total cholesterol > 240 mg%	30.6%
HT BP > 160/85 mm Hg	11.3%
Family history of coronary artery disease	11.3%
Obesity (> 20% of ideal body weight)	9.7%
Diabetes Mellitus	6.5%
Hyperuricemia (> 7 mg%)	3.2%

In studies done by Dwivedi, Girish, Chaturvedi, Sanju,et.al[55] which comprised of 70 young coronary artery disease patients ≤ 40 Years. There were 56 (80) males and 14 (20%) females, mostly (67.14%) belonging to 36 – 40 age group. More than half the patients (53%) were from low socio economic group.

61.42% were chronic smokers and all of them were males. 18.8% subjects gave history of premature coronary artery disease. Hypertension was detected in 51.42%. Obesity observed in 35.71%. Underweight was noted in 14% of subjects. 80.35% males and 91.66% females manifested upper segment obesity. Hypercholestrolemia was seen in 41.66%. 7.14% were diabetic. Family history of premature coronary artery heart disease in 42.8% and previous hypertension in 42.8% and central obesity (.9.166%) were three distinct risk factors in young female subset of patients.

DIWVEDI, GIRISH STUDY

Males	80%
Females	20%
Chronic smoker	61.42%
Premature coronary artery disease	18.8%
Hypertension	51.42%
Obesity	35.71%
Diabetes	7.14%
Family history	42.8%
Hpercholesterol	41.66%
Low Socio Economic Status	53%

Studies by Marty AK et al[56] showed the following data about the risk factors for acute myocardial infarction. Smoking was the most common risk factor present.

Smoking	66%
Hyperlipidemia	44%
Stress	40$
Hypertension	28%
Family history	28%
Diabetes Mellitus	18%

AIM OF THE STUDY

1. To study the clinical spectrum of myocardial infarction.
2. To study and assess the classic atherogenic risk factors taking into consideration both modifiable and non-modifiable factors.
3. To study the multiplicative effects of different combinations of the different Risk factors.
4. To thing more in terms of preventive cardiology than palliative cardiology.

MATERIALS AND METHODS

This case control study was conducted at the intensive cardiac care unit and medical wards of Chengalpattu Medical College Hospital, Chengalpattu during the period of September 2010 to November 2010.

- The study was done as a case control study retrospectively.
- The patients admitted with the clinical features of myocardial infarction with ECG changes suggestive of MI, elevated CKMB and SGOT, positive ECHO findings were taken as cases.
- The patient admitted with chest pain and the ECG, CKMB and SGOT,ECHO were not suggestive of myocardial infarction were taken as controls.
- Two hundred patients were examined and a detailed history with regard to risk factor analysis was made.
- The age group of less than 40 years were taken for study
- Both female and male patients were taken for study
- The patients were grouped into three categories according to the age 20 to 28, 29 to 34 and 35 to 40.
- The cases and controls were matched according to the age and sex.

OBSERVATIONS

In the study of clinical spectrum of myocardial infarction chest pain and its association with sweating, nausea, vomiting, breathlessness and palpitation were all taken into account and the percentage of each was studied.

Physical signs like hypertension, hypotension, raised JVP, edema, S_3, S_4 and wheeze were all looked for.

Smoking was considered as mild (upto 10 cig/day), moderate (10-15 cigarettes/day) heavy (> 15 cigarettes/day)

Hypertension was considered by documented history of hypertension on medication or BP > 140/90 mm Hg.

Diabetes Mellitus was considered either by documented history of treatment or with a fasting blood sugar level ≥ 126 mg and post prandial ≥ 200 mg.

Serum choleterol was done for all patients. A lipid profile was also performed.

Triglyceride value > 160 mg/dl and HDL < 40 mg/dl was taken into study.

LDL level should ideally be less than 130 mg/dl the LDL level was graded as <130, 130-160 & >160 mg/dl.

Questionnaire for personality traits was asked and patient's categorised as type A & type B.

Patients were grouped into three categories according to their age as 20-28, 29-34, 35-40.

A detailed family history, socioeconomic status, occupation, diet and lifestyle were obtained.

BMI was calculated by using wt/ht^2 and weight/hip ratio was calculated.

RESULT AND OBSERVATION

CLINICAL PRESENTATION – SYMPTOMS

92% of the patients with myocardial infarction had mild to severe chest pain. 62% presented with H/o excessive sweating, 30% presented with fatique and 22% associated with breathlessness.

The character of pain was assessed to be sharp stabbing with radiation in 42%. The radiation was mainly to the left arm, jaw, epigastric region. 2 patient presented with acute onset of giddiness, one patient with arm pain alone.

CLINICAL PRESENTATION

Symptoms	No. of Cases (Total 100)
Chest pain (angina mild to severe)	92
Chest pain with sweating	62
Radiation +	42
Fatigue	30
Breathlessness	14
Nausea and vomiting	9
Breathlessness without chest pain	8
Palpitation	6
Epigastric pain	5

CLINICAL PRESENTATION – SIGNS

Hypertension was the most common presenting sign. 56% of Patient were normal, Mitral regurgitation in 3%, S3 found in 8%, Crackles in 16%.

Signs	No. of Cases
Normal Cardiovascular Findings	56
Hypertension	42
Crackles	16
S3	9
S4	5
Hypotension	7
Wheeze	5
Associated MR	3
Apical shift	2
Raised JVP	2
Edema	1

NUMBER OF CASES ACCORDING TO KILIP'S CLASSFICATION

Killip gave a classification depend on the status of cardiac pump function, *estimated clinically.*

Class I - No sign of pulmonary or venous congestion.

Class II - Moderate heart failure as evident by rales at lung bases, S3 gallop, tachypnea or right sided heart failure signs.

Class III - Severe heart failure, pulmonary edema

Class IV - Shock with systolic pressure < 90mm Hg, Evidence of peripheral vasoconstriction, Peripheral cyanosis, mental confusion, oliguria.

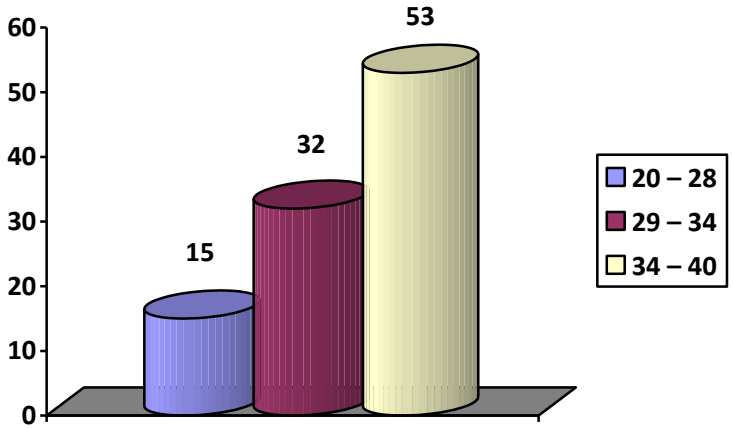
NUMBER OF CASES ACCORDING TO KILLIP'S CLASSIFICATION

Class	No. of Cases
I	84
II	11
III	4
IV	1

Age Distribution

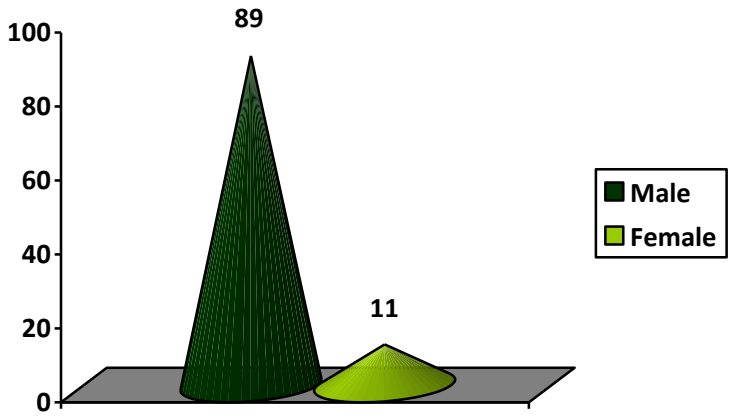
Age	No of person
20 – 28	15
29 – 34	32
34 – 40	53
Total	100

The youngest age is 22years who is a lorry driver,and heavy smoker for 5 years.



Sex Distribution

Sex	No of person
Male	89
Female	11
Total	**100**



Age sex ratio

Age	Male	Female
20 – 28	15 (16.9%)	0
29 – 34	30 (33.7%)	2 (18.2%)
35 – 40	44 (49.4%)	9 (81.8%)
Total	**89**	**11**

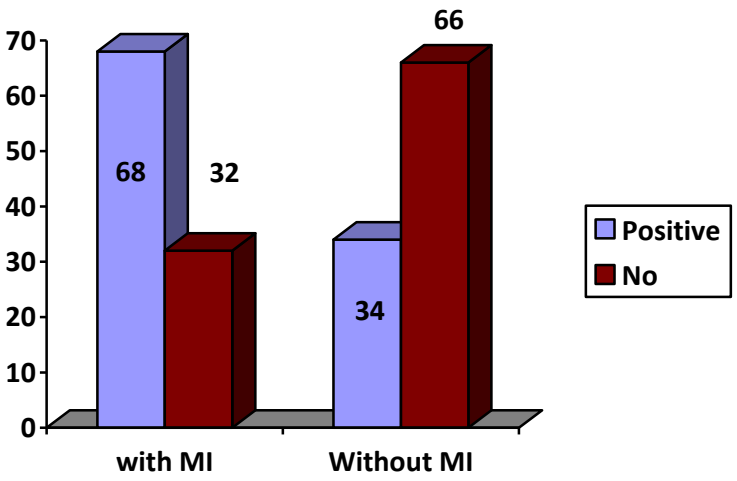
Females were somewhat elder to their male counter parts.most of them belong to 35-40 group.

Family history

	With MI	With out MI
Positive	68	34
No	32	66

OR = 4.13 (95%CI = 2.20 to 7.79)

Chi sq test = 23.13 p value = 0.0000 significant



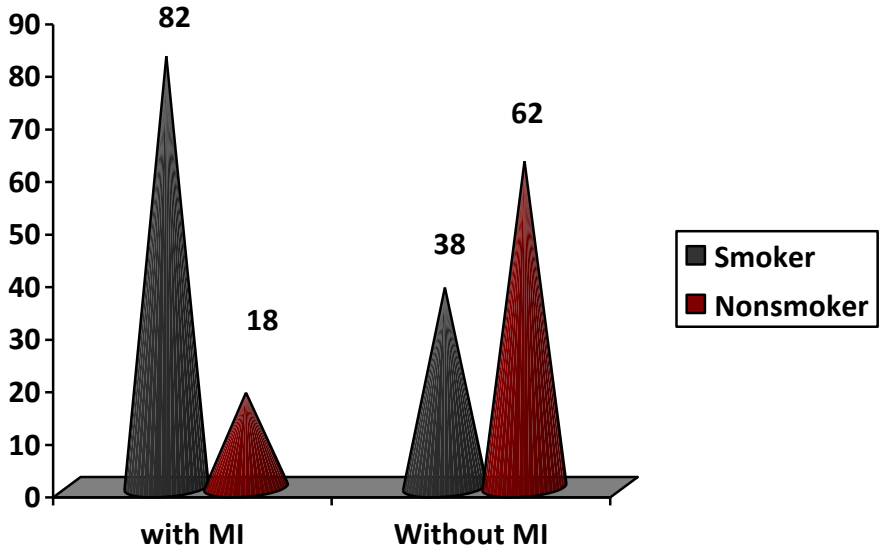
Family history was found in 68% cases. We have included coronary artery disease in all age groups. It is important to remember that positive family history of coronary artery disease in the young has genetic, environmental and life style components which ultimately culminate into young coronary episodes into the family.

Smoking vs MI

	With MI	With out MI
Smoker	82	38
Nonsmoker	18	62

OR = 7.43 (95%CI = 3.7 to 15.07)

Chi sq test = 40.33 p value = 0.0000 significant



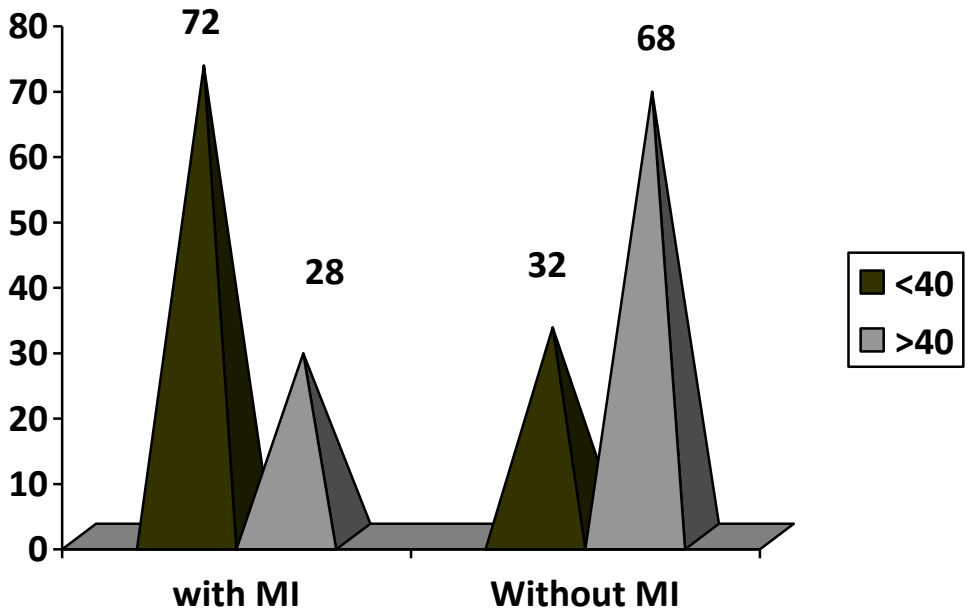
Smoking increases the risk of MI upto 8 folds.

HDL vs MI

	With MI	With out MI
<40	72	32
>40	28	68

OR = 5.46 (95%CI = 2.86 to 10.51)

Chi sq test = 32.05 p value = 0.0000 significant

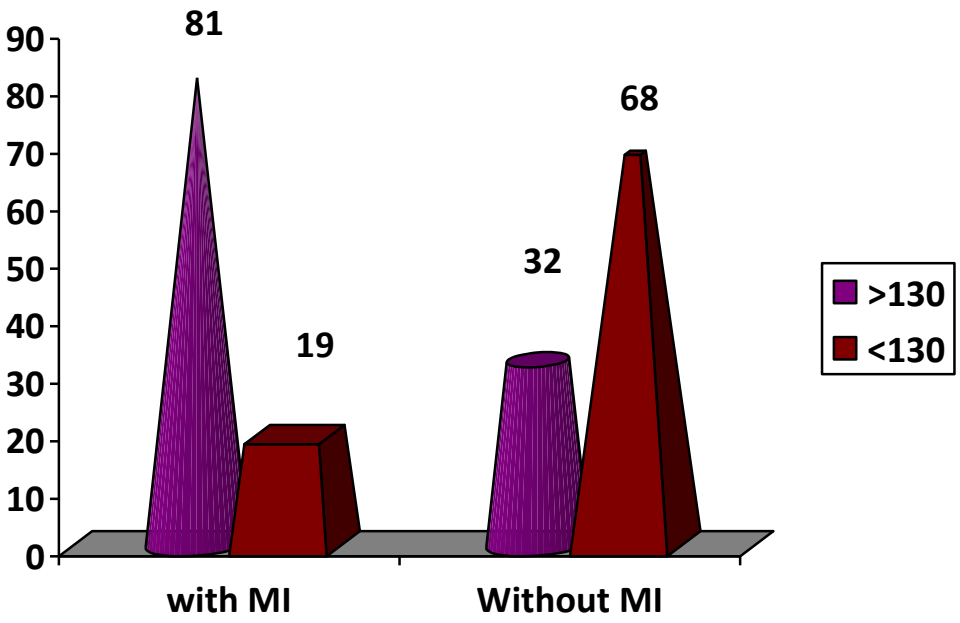


LDL vs MI

	With MI	With out MI
>130	81	32
<130	19	68

OR = 9.06 (95%CI = 4.5 to 18.42)

Chi sq test = 48.85 p value = 0.0000 significant

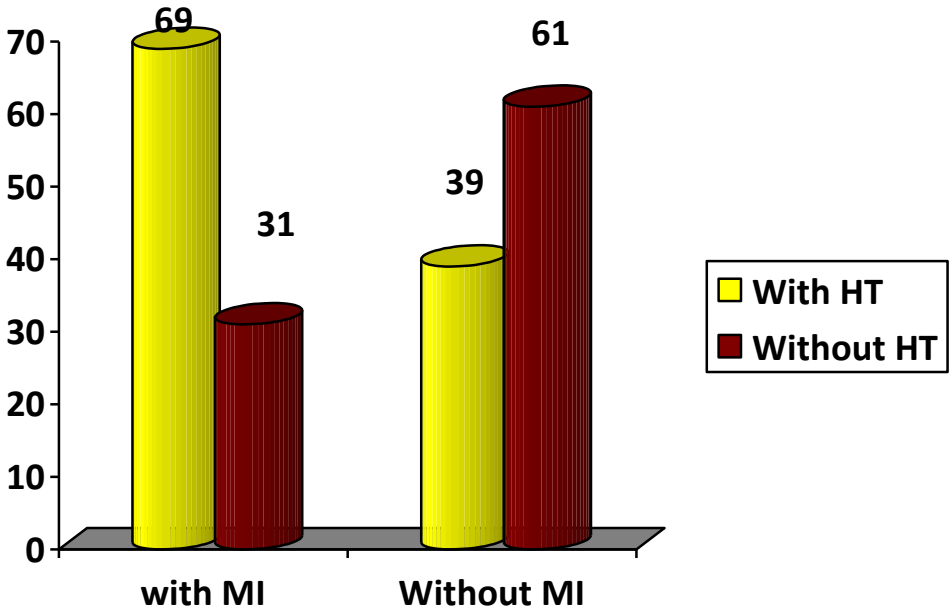


HT vs MI

	With MI	With out MI
With HT	69	39
Without HT	31	61

OR = 3.48 (95%CI = 1.87 to 6.53)

Chi sq test = 18.12 p value = 0.0001 significant



Obesity vs MI

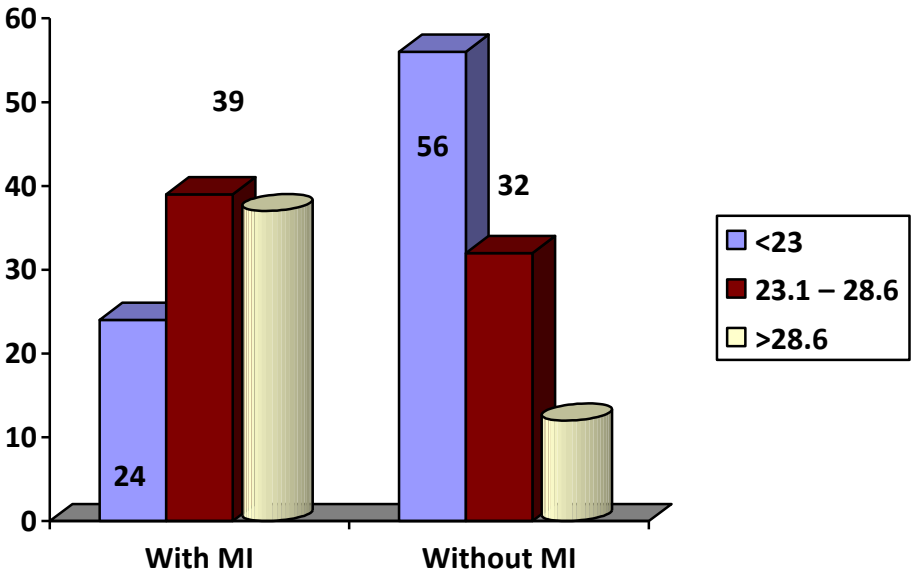
	With MI	With out MI
Obese	37	12
<28.6	63	88

OR = 4.31 (95%CI = 1.98 to 9.53)

Chi sq test = 16.89 p value = 0.0001 significant

	With MI	With out MI
<23	24	56
23.1 – 28.6	39	32
>28.6	37	12

Chi sq test = 26.25 p value = 0.0000 significant



SEDANTARY LIFE STYLE

Total No. of Patients with MI	H/O sedentary life style	Active Physical activity
100	**38**	**62**

Sedentary life style was noted in 38% of patients with myocardial infarction.

Waist hip ratio – male vs female

	Male	Female
<desired level	29	2
>desired level	60	9

Desired level for male < 0.9

Desired level for female < 0.8

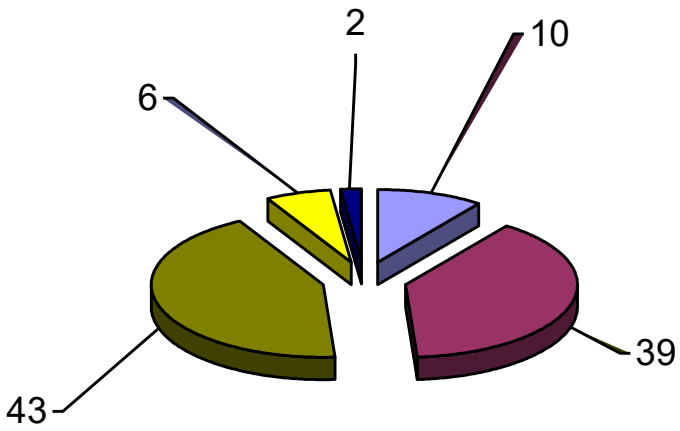
OR = 2.17 (95%CI = 0.4 to 15.63)

DM vs MI

	With MI	With out MI
DM+	17	8
DM-	93	92

OR = 2.1 (95%CI = 0.81 to 5.62)

Economic Status of Patients under Study



49% of patients belonged to low socio economic group, while 43% came from middle income group and only 8% were from upper group. Existence of coronary artery disease in the lower socioeconomic group of people in the polis of India is a fact, which is being observed in this country. The scenario has changed considerably since the 70's, when low socio economic group of people in rural and semi urban India used to do a lot of physical labour and hard work and incidence of ischemic heart disease was reported to be low in such cases. These days many of the young able bodied people migrate to neighboring metropolis and adopt faulty life styles which make them prone to coronary artery disease.

80% of young coronary artery disease subjects were chronic beedi smokers. All of them were males and they come from low socioeconomic and middle income segment of the society. Another interesting observation was the

fact that only 35% males had smoking as the only conventional risk factors, however many of them had three of four major risk factors.

SOCIO ECONOMIC STATUS OF PATIENTS

SES	No. of patients with MI
Poor	10
Lower middle	39
Middle	43
Upper middle	6
Rich	2

It is also well known that combination of major risk factors like smoking, hypertension and dyslipidaemia in an individual exerts a multiplicative effect on coronary artery disease. The risk increases as much as 9-16 times when all three risk factors are present.

Psychosocial stress :

- Psychosocial stress was found to be present in 38% of the patients with myocardial infarction. They were all Type A personalities with an evidence of stressful environment.

Diet :

- Dietary history showed that myocardial infarction was more common in non-vegetarians than vegetarians.

AREAS OF INFARCTION

In our study the most common site of infarction was found to be anterior wall accounting for 47% of cases.

INFARCT SITE	CASES
Extensive ant wall infarct	47
Anteroseptal	24
Inferolateral	8
Inferior + Right Ventricle	5
Anterior + Inferior	4
Inferior + Posterior	8
Anterolateral	4

TYPE OF INFARCTION

64% of patients admitted with myocardial infarction had a Q Wave in ECG.

Q Wave	64%
Non Q Wave	36%

SUMMARISING THE RESULTS

Males	89%
Smoking	82%
Hypercholesterolemia	79%
Family History	68%
Low socioeconomic status	49%
Hypertension	39%
Sedentary lifestyle	38%
Psychosocial stress	38%
Obesity	37%
Diabetes Mellitus	15%

COMBINATION OF RISK FACTORS IN MI

HT + Obesity + DM + Hypercholesterolemia	5
Hypercholesterolemia + Obesity + DM	10
Obesity + Hypertension	15
Obesity + Diabetes mellitus	7
Obesity + Hyper cholesterolemia	21

Some had clustering of several coronary risk factors.

DISCUSSION

This Hospital based study of clinical spectrum and risk factors in the young myocardial infarction patients the commonest symptoms was chest pain and the commonest sign was hypertension. Risk factor analysis showed that smoking, hypercholestrolemia, positive family history, hypertension, obesity were frequently associated with young myocardial infarction patients. We are comparing the results of the study with the previous studies.

PRESENTING SYMPTOMS AND SIGNS

In our study most common presenting symptom was chest pain. Other anginal equivalence accounted for 8% of presentation. In our study 15% of the patient were diabetics. Almost all the diabetic patient presented with anginal equivalents. Similar observations have been made in other studies also i.e. **Mrgolin JN, Kinnel WB[62], Fienleich M, et al. (23%) Clinical features of recognized MI silent and symptomatic in AMJ. Cardial 1973 : 32:1, PA Tambyah et al.**[50]

The most common cardiovascular sign was hypertension (42%) similar observations have been made in other studies also i.e. **Marty[56] AK Das AK et al. (41%), Nitter Haugh[53] et al (24%), Dwivedi[55] et al (51.42%) studies.**

RISK FACTORS

Risk factors in young coronary artery disease: Indian Scenario

	Tambya 1999 n=32	Dolder 1985 n=240	Rhotak 1978 n=85	Kaul MD 1986 n=105	Gupta R 1994	PK Biswas 1994	Dwiv 2000 n=70	Kellelly 1982 N=145	As per the present study
Male : Female	NR	NR	NR	9:1	NR	NR	4:1	9:1	9:1
Family history %	20	12	55	28	NR	13.3	42.8	31	68
Smoking %	84	80	17	76	42	56.4	61.42	80	82
Hyper-tension %	19	15	15	32	13	11.3	51.42	11	39
Obesity %	15	NR	15	NR	10	9.7	35.71	27	37
Diabetes Mellitus %	16	10	NR	5	4	6.5	7.14	NR	15
High cholesterol %	56	46	30	36	53	30.6	41.66	70	81

NR – not recorded

SMOKING :

In our study 82% of the patients were smokers all of them are males. The probable reason being the atherosclerotic process accelerated by enhanced oxidation of LDL-C and reduced the levels of HDL-C. Smoking also impairs endothelium, increases inflammatory markers and fibrinogen, causes platelet aggregation and increases monocyte adhesion to endothelial cells. In earlier studies too similar observations have been made i.e. **Kaul MD et al[48] m 1986 (76%), PA. Tambyah et al. [50] (1999) 84% Dolder M et al[51] (80%) Swedarsen et al[52] 1987 (79%) Dwivedi[55] et al. (61.42%)**

Hypercholestrolemia:

In our study 81% of patients were found to have dyslipidemia. Hypercholestrolemia accelerates the atherosclerotic process. In addition hypercholestrolemic patients who are associated with obesity, insulin resistance also produces a prothrombotic state due to increased level of PAI – I and Fibrinogen Similar observations have been made in other studies also i.e. **Dolder et al (54%)[51] (68%) 1987, PK Biswas A Dasbiswas S Roy[54] et al (30.6%) in 1994, VNS[61] study (92.8%) in 1971 BOM61 study (70%) in 1970, Knennelly Bm etal study[58] (70%)**

Family History :

In our study 68% of the patients were found to have positive family history. The genetic factors contribute to the risk of developing ischemic heart disease. The risk is as high as 40%. Common deletion polymorphism of angiotension conveting enzyme (ACE) gene associated with increased level of ACE adds to the risk of coronary artery heart disease. Genetic factors such as LDL receptor factor V Leiden are also other genetic markers. In earlier studies too similar observation have been made i.e. **Marty[56] AK Das AK et al (28%), PK Biswas A Dasbiswas S Roy[54] et al (11.3%) in 1994, Dwivedi[55] et al (42.8%) in 2000, VNS60 study (25%) in 1971, Rhotak[61] study (55%) in 1978, Kennelly BM et al[58] (31.%)**

Age Sex Ratio:

In our study males were 89% and females were 11% (Male & Female ratio is 6.69:1) Significantly major risk factors like smoking, psychosocial stress and hypertension were among men as compared to women. In females

the risk of developing coronary artery disease started 10 years later than male. This is presumably due to hormonal factors. The most common age groups affected were between 36 to 40 years. This denotes increasing incidence of myocardial infarction with increase in age. Similar observations have been made in other studies also i.e. **Dwivedi[55] et al (4:1) in 2000, Bikanes[61] study (8:1) in 1994, VNS[61] study (8:3:1) in 1971, old Delhi[61] study (9:1) in 1986.**

Hypertension:

In study 39% of the patients were found to have hypertension. The probable reason being the accelerated atherosclerosis, increased left ventricle wall stress, left ventricle tension and stroke work. Other reasons like left ventricular hypertrophy, abnormal coronary flow reserve and abnormal vasomotor response and micro vascular dysfunction. Similar observation have been found in other studies also i.e. **Marty[56] AK Das AK et al (28%), Nitter Haugh[53] et al (24%) Dwivedi[61] et al (51.42%) in 2000. Kaun MD et al 32%[68].**

Psychosocial stress :

In our study 38% of patients were found to have psychosocial stress. The probable reason is depression, job stress, social isolation, Type A personality are some of the factors contribute to an elevated risk of Coronary artery heart disease, although further data are needed to confirm this relationship. Similar observations have been made in other study also. I.e. **Marty[56] AK Das AK et al (40%)**

Obesity :

In our study 37% of patients were found to have obesity. Obesity is an important risk factor in the development of coronary heart disease. Obesity is associated with increase in total and central blood volumes, cardiac output and left ventricular filling pressure. When the additional effects of hypertension and glucose tolerance are added, the adverse impact of obesity is even more evident. Obesity especially central obesity is associated with an atherogenic lipid profile. Similar observation have been made in other studies also i.e. **Dwivedi[55] et al (35.71%) in 2000, PK Biswas A (9.7%), VNS[61] study (35.7%) in 1971, old Delhi[61] study (11%) in 1986, Rhotak[61] study (15%) in 1978, BOM[61] study (23.5%) in 1979, Kennely Bm etal[58] (77%)**

Diabetes Mellitus :

In our study 15% of patients were found to have diabetes. The reason being it impairs endothelial and smooth muscle function and appears to increase leukocyte adhesion to vascular endothelium, a critical early step in atherogenesis. And also insulin resistance also produces a prothrombotic state due to increased level of PAI – I and fibrinogen. Similar observations have been made in other studies also i.e. **Marty[56] AK Das AK et al (18%), PK Biswas A (6.5%), VNS[61] study (17.8%) in 1971, old Delhi[61] study (3%) in 1986, Dwivedi[55] et al (7.14%) in 2000, Chennai[61] study (18%) in 1991. Dolder m et al. 10%[51]**

CONCLUSION

The following are conclusions that could be inferred from the study on the clinical spectrum and risk factors among young myocardial infarction.

- The most common symptom was chest pain.
- The most common cardiovascular sign was hypertension.
- Most of the patients belonged to Killip's classification I.
- Most common age group affected was between 36 to 40 years showing that risk of myocardial infarction increase proportionately with increasing age.
- Males were commonly affected especially in the younger age group. Significantly major risk factors like smoking, psychosocial stress, and hypertension were also evident among men as compared to women. These factors along with hormonal factors contribute the higher proportion of myocardial infarction in young males.
- Females showed an increased risk of myocardial infarction towards the later stages of life presumably due to hormonal factors.
- Risk factors analyses proved that smoking was the single most important risk factor for myocardial infarction.
- Majority of patients with myocardial infarction had dyslipidemia.
- Family history of myocardial infarction is an important risk factor contributing to myocardial infarction in young individuals. This is probably due to an inter play of both genetic and environmental factors.

- Hypertension was closely related with risk of myocardial infarction.
- The obesity was an important risk factor in young myocardial infarction patients probably due to the increasing incidence of sedentary life style.
- The incidence of diabetic mellitus in young myocardial infarction patients was comparatively less than the incidence of other risk factors.

SUMMARY

Chest Pain is the cardinal symptom in young myocardial infarction. Breathlessness, sweating, radiation, vomiting, palpitation, sweating are common features symptoms. Signs of hypertension, S3, S4, crackles, were commonly seen. On few occasions infarction also occurred in the absence of physical signs or symptoms.

Key factors influencing the development of myocardial infarction were non modifiable risk factors such as age, sex, family history. Smoking is probably the single most important risk factor. There is a strong relationship between cigarette smoking and coronary artery heart disease in young individuals. The risk is also closely related to plasma LDL cholesterol, and inversely related to HDL cholesterol concentration. Obesity particularly central or truncal is an independent risk factor. Additional risk factors such as diabetes, physical inactivity added to the adverse impact.

The effect of risk factors is multiplicative rather than Additive. Thus people with the combination of risk factors (smoking, hypertension, diabetes) have the greatest risk of developing myocardial infarction.

Prevention can aim at modifying the risk factors like cessation of smoking, reduction of weight, reduction of salt intake, dietary changes, increase physical activity and control of psychosocial stress. This will have a tremendous impact in reducing the incidence of myocardial infarction in the young.

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CLINICAL PROFORMA

Name :	Height :
Age :	Weight :
	BMI (Wt/Ht in m^2)
Sex :	
Occupation :	Waist/Hip Ratio
Income/Year	Educational Status :

Clinical presentation of illness

Chest pain – Characters :

Compressive, Retrosternal, Consistent, Radiating to left arm or root of the neck or back of the chest,

Angina equivalent -

H/o nausea, vomiting, epigastric pain, jaw pain neck pain, left arm pain.

Profuse sweating, fatigue, breathlessness, palpitation, syncope

Time of onset of symptoms :

Admission time : Therapeutic Window.

Risk Factor Identified :

1. Smoking : Beedies/Cigarettes smoking / Others
   No. of Cigarettes/day No. of years of smoking :
   Other forms of tobacco : Nasal snuff / tobacco snuff / Chewing
2. Hypertension : How many years :
   Under treatment – Yes/No Regular / Irregular Rx :
3. Diabetes Mellitus : IDDM/NIDDM (How many years)
   Treatment : Regular Rs/Newly diagnosed
4. Family H/o ischemic heart disease
5. Obesity
6. Alcohol consumption : Little/Moderate/High
   Occasional uses/regular uses
7. Hyperlipidemia : No. of years Treatment :

III. OCCUPATIONAL HISTORY :

IV. DIET HISTORY

Vegetarian/Non Vegetarian High carbohydrate diet
Salt intake High fat intake
Coffee/Tea
Living Condition : Nuclear Family / Joint family /Alone
Hut / Tiled House / Independent / Flats
Hygiene

Menstrual history :

Menopause : Years since menopause

Sexual history :

Frequency of intercourse/extramarital relationship

Personality :

Type A	Type B
Never	Casual about appointment
Very competitive	Not competitive
Always rushes	Never
Impatient when waiting	Can wait
Tries to do many things at once	Take things one at a time
Expresses feelings	Does not
Ambitious	Not Ambitious

General Examination

Conscious : Orientation :

PR BP RR JVP

Pallor Cyanosis

Systemic Examination (CVS)

Nature of apical impulse Abnormal systolic pulsation

S_1, & S_2 intensity S_3 / S_4

Murmur of MR/VSD secondary to septal rupture

Pericardial friction rub

(RS) Crackles : Wheeze :

INVESTIGATIONS

I.	Blood	1.	Hb		
		2.	Sugar		
			Urea		
			Cretinine		
			Electrolytes		
		3.	Cardiac enzymes	-	Troponin - T
					Cpk mB
					SGOT
		4.	Liquid profile		
2.	ECG				
3.	Chest X-ray				
4.	ECHO				

KEY NOTES FOR MASTER CHART

Sl. No	-	Serial Number
Sx	-	Sex

Clinical spectrum – Symptoms and signs

CP	-	Chest Pain
RA	-	Radiation
S	-	Sweating
N/V	-	Nausea/Vomiting
DWOC	-	Dysponea Without Chest Pain
DWC	-	Dysponea With Chest Pain
F	-	Fatigue
P	-	Palpitation
EP	-	Epigastric Pain
H	-	Hypotension
RJ	-	Raised JVP
PE	-	Pedal Edema
AS	-	Apical Shift
MR	-	Mitral Regurgitation
R	-	Rales
W	-	Wheeze

Risk Factors

SM	-	Smoking
HT	-	Hypertension
DM	-	Diabetes Mellitus
FH	-	Family History
OB	-	Obesity
LDL	-	Low Density Lipoprotien
HDL	-	High Density Lipoprotein
LSES	-	Low Socio Economic Status
SL	-	Sedentary Life
PSS	-	Psycho Social Stress

MASTER CHART

Sl.No	Age	Sx	CLINICAL SPECTRUM - SYMPTOMS AND SIGNS																		RISK FACTORS									
			CP	RA	S	N/V	DWOC	DWC	F	P	EP	H	RJ	PE	AS	S3	S4	MR	R	W	SM	HT	DM	FH	OB	LDL	HDL	LSES	SL	PSS
1	38	M	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	+	+	-	+	+	-
2	28	M	+	+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	+	+	+	+	-	-
3	34	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	+	+	-	-	-
4	39	M	+	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	-	+	+	+	-	+
5	40	M	+	+	+	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-	+	-	-	+	+	+	-	-	+	-
6	34	M	+	+	+	-	-	-	+	-	-	+	+	+	-	-	-	-	+	-	+	-	-	-	+	+	-	+	-	+
7	39	M	+	+	+	+	-	+	-	-	-	+	-	-	-	-	-	-	+	-	+	-	+	+	+	+	-	-	+	-
8	33	M	+	-	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	+
9	39	M	+	+	-	-	-	-	+	-	-	-	-	-	-	+	-	-	+	+	-	+	-	+	+	+	+	+	+	-
10	38	F	+	-	+	-	-	-	-	+	-	+	-	+	-	-	-	-	-	-	-	-	-	+	+	+	-	-	+	+
11	28	M	+	+	-	-	+	+	-	-	-	+	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	+	-	-
12	39	M	+	+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	+	+	-	+	+	+
13	33	M	+	+	-	-	-	-	-	-	+	-	-	-	-	+	-	-	+	-	+	-	-	-	-	+	-	-	-	-
14	34	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	+	-	+
15	38	M	+	-	-	-	-	-	+	-	-	-	-	-	-	-	+	-	+	-	+	+	-	+	+	+	-	-	+	-
16	33	M	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	-	-	+
17	39	M	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	+	+	-	-	+	-
18	30	M	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	+	+	+	-	+	-	-	-	+
19	40	M	+	+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	+	-	-	-	+	-
20	39	F	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	-	+	+
21	37	M	+	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	+	-	-	-	+	+
22	29	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	-	-	+
23	34	M	+	-	+	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	-	-	-	+
24	40	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	+	-	-	-	-	-
25	33	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	+	-	-

MASTER CHART

Sl.No	Age	Sx	CLINICAL SPECTRUM - SYMPTOMS AND SIGNS																		RISK FACTORS									
			CP	RA	S	N/V	DWOC	DWC	F	P	EP	H	RJ	PE	AS	S3	S4	MR	R	W	SM	HT	DM	FH	OB	LDL	HDL	LSES	SL	PSS
26	33	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	-
27	29	M	+	+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	-
28	36	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	-	+	+	+	-	-
29	32	M	+	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	+	-	-
30	39	M	+	-	+	+	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	+	+	-	-	-
31	34	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	+	-	-
32	35	M	+	-	+	-	-	-	-	-	-	+	-	+	-	-	-	-	+	-	+	-	-	+	-	+	-	-	-	-
33	33	M	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	-
34	39	M	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	-	-	-	+	-	-
35	39	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	+	+	+	-	+	+
36	37	F	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	+	+
37	39	F	+	+	+	+	-	-	-	-	-	-	-	-	+	+	-	+	+	+	-	+	+	-	+	+	+	-	+	+
38	27	M	+	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	-
39	34	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-
40	28	M	+	-	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	+	-	-
41	31	M	+	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	+	+	-	-	+	-	+	+	-	-	-
42	38	M	+	-	-	-	-	-	+	-	-	+	-	+	-	-	-	-	-	-	+	-	-	+	+	+	+	+	+	+
43	30	M	+	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	-	-	-
44	37	M	+	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	-	+	-	+	-	-
45	33	M	+	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	-	-	-
46	39	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	+	+	+	-	+	+
47	32	M	+	+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	-
48	38	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	+
49	33	F	+	+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	+	+	+
50	38	F	+	+	-	-	-	+	+	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	+	-	-	+	+

MASTER CHART

Sl.No	Age	Sx	CLINICAL SPECTRUM - SYMPTOMS AND SIGNS																		RISK FACTORS									
			CP	RA	S	N/V	DWOC	DWC	F	P	EP	H	RJ	PE	AS	S3	S4	MR	R	W	SM	HT	DM	FH	OB	LDL	HDL	LSES	SL	PSS
51	34	M	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	+	-	-
52	28	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	+	-	-
53	38	M	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	+	+	+	-	+	+
54	39	M	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	+	+	-	+	+	+
55	33	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	+	-	-
56	39	F	+	-	+	-	-	-	+	-	-	-	-	-	+	-	-	-	+	-	-	+	-	+	+	+	+	-	+	+
57	39	M	+	+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	+	-	-
58	34	M	+	+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	+	-	-
59	38	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	+	+	+	+	-	+	+
60	39	M	+	+	-	-	-	+	-	+	-	-	-	-	-	+	+	-	+	-	+	+	-	-	+	+	-	+	+	-
61	38	F	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	+	-	+	+	-
62	37	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	-	-	-
63	33	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	-	-	-
64	39	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	+	-	+	+	+
65	38	F	+	-	+	-	-	-	-	-	-	-	-	-	-	+	+	-	+	+	-	+	-	+	-	+	+	+	-	-
66	34	M	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	-	+	-	-	-	-
67	29	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	+	-	-
68	34	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	-	-	-
69	31	F	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	+	-	-
70	32	M	+	+	+	-	-	+	-	-	-	+	+	+	+	+	-	+	+	-	+	-	-	-	-	+	+	+	-	-
71	38	M	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	+	-
72	25	M	+	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	+	+	-	+	+	-	-	-
73	34	F	+	+	+	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	+	+	-
74	29	M	+	+	+	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	+	-	-	+	-	+	+	+	-	-
75	39	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	+	+	-	-	+	-

MASTER CHART

Sl.No	Age	Sx	CLINICAL SPECTRUM - SYMPTOMS AND SIGNS																		RISK FACTORS									
			CP	RA	S	N/V	DWOC	DWC	F	P	EP	H	RJ	PE	AS	S3	S4	MR	R	W	SM	HT	DM	FH	OB	LDL	HDL	LSES	SL	PSS
76	38	M	+	+	+	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	+	-	+	+	+
77	34	M	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	+	+	+	-	+	-	+	-	-
78	40	M	+	+	+	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	-	+	-	-	+	+	+	-	+	+
79	39	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	+	+	-	+	+	+
80	39	M	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	+	+	+	-	+	+
81	29	M	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	+	+
82	34	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	-	-	-
83	31	M	+	-	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	-	-	-
84	39	M	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	-	-	+	+	+	+	+	+	+
85	40	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	-	+	-
86	38	F	-	-	-	-	+	-	-	-	-	-	-	-	+	+	-	+	+	-	-	-	-	+	+	+	+	+	-	+
87	33	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-
88	33	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	-	-	-	-
89	39	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-
90	34	M	+	+	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	+
91	29	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	+	-	-	-	+	-
92	30	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	-	-	+
93	37	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	+	-	-
94	40	M	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-	+	-	-	+	-	+	-	+	+	-	-	+
95	28	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	+	-	-
96	33	F	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	+	+	+	+
97	38	M	+	+	+	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	-	-	-
98	31	M	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	+
99	27	M	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	-	-
100	38	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	+	+	-	-	+	-